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Toxicologist Argues TCE Non-Cancer Risk Likely To Drive Many Cleanups

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An environmental consultant who helped industry gain a review from an alliance of risk assessment experts of EPA's policy decisions regarding the ubiquitous solvent trichloroethylene (TCE) is arguing in a new paper that the non-cancer risk level in the agency's TCE risk assessment will become a driving factor in many cleanups despite a lack of EPA guidance on the issue.

Rod Thompson, of Risk Options, LLC, and the Alliance for Site Closure (ASC), argues that the practical application of EPA's TCE reference concentration (RfC) -- or the amount of the substance EPA anticipates can be inhaled daily over a lifetime without causing adverse health effects -- of 2 micrograms per cubic meter (ug/m^3) will create new environmental challenges.

EPA finalized a new RfC and reference dose -- or amount of the substance that can be ingested daily over a lifetime without causing adverse health effects -- for TCE in September 2011, significantly strengthening its risk values for the contaminant.

Cleanup challenges will arise because for many states the RfC will lead to non-cancer standards that are stricter then the cancer ones, and so non-cancer levels will drive site closure decisions, a shift that takes flexibility away from decision-makers because standards to prevent non-cancer health effects are based on a threshold level rather than a range of risk levels available for preventing cancer, Thompson says in the paper. The paper will be presented to the panel of risk assessment experts examining EPA's TCE policies.

The need to use a threshold level for cleanup decision-making also raises questions about how to interpret sampling results close to the RfC level, he says.

"These TCE science and science policy considerations create new questions that must be addressed if States are to make sound health protective decisions during risk assessment investigations and remedial actions," Thompson states.

The effect that the RfC will have on risk assessments, as well questions over short-term exposure levels to protect against cardiac birth defects, prompted ASC in July to petition the Alliance for Risk Assessment (ARA) to review EPA's recent TCE policy and provide guidance to better understand the risk of non-cancer effects. In the request, which Thompson co-authored, ASC also asked that the review include an evaluation of the margin of safety included in the RfC, and to assess whether EPA derived the RfC in accordance with proper principles.

ARA accepted the proposal in August, and is currently scheduling conference calls to address the issues raised in the ASC proposal.

Weight Of Evidence

In addition to raising concerns about EPA's method of deriving the RfC, and how the non-cancer number could alter cleanups, Thompson revisits industry objections to EPA's use of a 2003 study that showed cardiac birth defects in rats as a basis for the RfC. After reviewing the reasons for and against using Paula D. Johnson's study as a factor in setting health standards for humans, Thompson says, "Overall weight of evidence for TCE induced fetal heart malformations by the inhalation route of absorption is questionable and not conclusive."

How to set short-term exposure levels for TCE to protect against cardiac birth defects is a question EPA headquarters is currently considering at the request of EPA Region IX and industry. Earlier this year, Region IX proposed an interim Removal Action Level (RAL) of 15 ug/m^3, derived from EPA's RfC, to protect construction workers from exposures that could cause cardiac birth defects. The workers are renovating offices near the Middlefield-Ellis-Whisman Superfund site in Mountain View, CA, for companies including Google, Inc. The interim RAL was proposed for buildings where workers are making holes in building foundations, which increases risk of vapor intrusion. Vapor intrusion occurs when toxic vapors rise from underground contamination into basements or buildings, through dirt floors, cracked foundations or other pathways.

While EPA headquarters is reviewing whether the RAL was appropriately derived from the RfC, industry officials have said the science relative to potential cardiac birth defects is too uncertain to require strict cleanup levels, that the method for crafting the limit is at odds with agency policy, and that the limit is orders of magnitude stricter than similar levels crafted by other agencies.

A source familiar with the issue has said industry concerns might be driven by fears the proposed limit could open the door to strict new cleanup requirements and bolster future personal injury and worker protection claims that might be brought against private and federal responsible parties at the hundreds of sites nationwide where the chemical is present.

In addition to the short-term exposure issue, the practical application of the RfC raises basic questions of how to assess sites, Thompson argues, saying that regulators and others who have to close sites to facilitate property transactions need to better understand the risks and effects of TCE exposure.

When states make site closure decisions, they calculate acceptable exposure levels for cancer and non-cancer endpoints, and then regulate to the stricter of the two levels, which traditionally has been the cancer endpoints.

In his paper, Thompson also weighs whether cardiac birth defects should have been included in the IRIS assessment as one of the health hazards for TCE. The question returns to industry concerns raised in 2009 when EPA floated a draft of its Integrated Risk Information System (IRIS) assessment for TCE and industry sources criticized the inclusion of the Johnson study, saying it was not a reliable study.

In questioning whether the 2003 study showing cardiac birth defects in rats should have been used to set the RfC, Thompson cites the IRIS assessment, which acknowledges "weakly suggestive, but overall consistent, epidemiologic data" as a factor, along with animal studies, for concluding TCE poses a potential risk of cardiac birth defects.

EPA cites two main human studies to support the risk of cardiac birth defects. One was conducted by the Agency for Toxic Substances & Disease Registry (ATSDR) in an area where there was significant vapor intrusion of TCE, and a second examined exposures in the Milwaukee, WI, area.

Interpretation Of Results

Thompson notes that ATSDR said interpretations of its results "must be made with caution due to the extremely small number of infants born with these birth defects." And in the second study of children with congenital heart defects, Thompson says the increased risk of congenital heart defects was seen only in children of mothers over the age of 38, while children of women under 38 showed no increased risk.

In arguing that evidence that TCE causes cardiac birth defects in humans is weak, and that the study should not be used, Thompson says numerous other animal studies have not shown cardiac defects. In addition, no inhalation animal studies have shown evidence of cardiac defects, Thompson says, and the animal studies, which did show cardiac defects were in one lab, and that additional studies designed to show similar results failed, even when conducted in the same lab.

"The inability to duplicate the Johnson et al. (2003) results is significant," the paper says. "Here the fetal heart malformation critical effect does not appear to be reproducible outside one testing facility and there is no evidence that this effect is even expressed in inhalation studies."

The final concern Thompson addresses in the paper is the margin of safety EPA included in its derivation of the RfC from the 2003 study. In the petition Thompson co-authored for ASC requesting the ARA review, he says EPA considers a range of possible values for the RfC and chose one at the low end of the range. Before that choice, EPA's derivation of possible RfC values had involved policy choices that "appear to be new or more conservative" then the usual methods, the request said.

In the paper, Thompson says the process used physiologically based pharmacokinetic modeling to determine the dose an animal absorbed from the dose it was given. The dosing was oral, and Thompson notes "no inhalation exposure data were used in the RfC analysis and inhalation toxicity is extrapolated from oral dosing."